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Near fatal ingestion of oil of cloves

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Abstract

A case of ingestion of oil of cloves is presented, which resulted in coma, fits, a coagulopathy, and acute liver damage. This is not unlike the syndrome produced in the late stages of a substantial paracetomol overdose, and a similar treatment regimen is proposed.

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Clove oil is an essential oil that is freely available on an 'across the counter' basis, having been used as a remedy for toothache for over 300 years. The earliest known reference for using the oil of cloves is in the *Practice of Physic*, published in French in the 1640s and translated into English in 1687.[1] The oil is obtained by distillation from the dried flower buds of the *Eugenia caryophyllata* tree of the myrtle family and contains 70–90% eugenol with a number of impurities. Eugenol is also used in the food and pharmaceutical industries and in a wide variety of herbal medicines.

Case report

The patient, a 2 year old boy, presented one hour after drinking between 5 and 10 ml of clove oil. Initial examination showed the patient to be distressed and crying, albeit a little drowsy, but otherwise normal. However, within three hours he had deteriorated into a deep coma with a marked acidosis. Eight and a half hours after ingestion the patient had a generalised seizure treated with intravenous diazepam. Also at this time the patient had an unrecordable blood glucose concentration; this was treated with an intravenous bolus of dextrose followed by a dextrose infusion.

Twenty four hours after ingestion the patient was still unconscious and showing laboratory evidence of deteriorating liver function. His international normalised ratio (INR) was 6·51 and alanine aminotransferase was 1549 IU/l (normal range <34 IU/l). At this point his haemoglobin concentration was 131 g/l with a platelet count of 56×10^9/l. A repeat INR after vitamin K was 10·48 and fibrin degradation products were >250 IU/l (normal <250). Fresh blood was aspirated from his nasogastric tube and so a primary diagnosis of disseminated intravascular coagulopathy (DIC) was made, although the deranged clotting could have been solely due to hepatic dysfunction. In order to treat the presumed DIC, the patient was started on heparin 200 IU/hour, fresh frozen plasma, and fibrinogen.

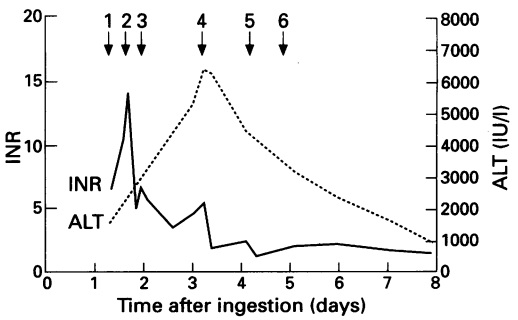
From the third to fourth days after ingestion the patient was treated for DIC with the administration of fresh frozen plasma, heparin, antithrombin III, protein C and factor VII, guided by the INR and alanine aminotransferase results (see figure). At the same time it was first noted that the patient's right optic disc margin was blurred. This coincided with a further deterioration in his conscious level, corresponding to the continuing deterioration of his liver function tests. There were no other signs of raised intracranial pressure.

During the fifth day the patient's neurological state gradually improved such that he was showing signs of waking and on day 6 he was fully conscious. From that time on he continued to improve and eventually made a full recovery.

Discussion

Numerous essential oils are available freely with little or no control on distribution. With the increasing popularity of aromatherapy and other natural remedies the prevalence of essential oils in the community is also increasing. Although little is known of the toxic effects of these oils, it is known that essential oils as a group can cause coma, fits, inhalation pneumonitis, localised anaesthesia,[2] respiratory depression, renal failure, hypoglycaemia, and lactic acidosis.[3] In addition to some of the above our patient also suffered from DIC and liver damage (not previously reported in conjunction with clove oil). Despite such toxic effects the warnings on the bottles are very general with 'keep all medicines out of the reach of children' being on the bottle in our case and no reference to the potentially very toxic effects.

There are a number of similarities between paracetomol and eugenol poisoning. In normal doses paracetomol is metabolised to glucuronide and sulphate conjugates, but in overdose this pathway is saturated and



INR and alanine aminotransferase (ALT) and treatments given. (1) Vitamin K 5 mg intravenous; (2) fresh frozen plasma, heparin, and fibrinogen (1 g); (3) antithrombin III 1050 U, protein C 880 U; (4) antithrombin III 525 U, protein C 880 U, factor VII 550 U, neomycin, and lactulose; (5) antithrombin III 525 U, protein C 880 U, factor VII 500 U; (6) vitamin K 5 mg intravenous.

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alternative pathways are utilised, a process aided by treating patients with acetylcysteine or methionine. Eugenol is also metabolised to glucuronide and sulphate conjugates by the liver, and has been shown to be hepatotoxic in rats with cell death occurring in >85% of rat hepatocytes exposed to eugenol after five hours; this effect is completely prevented by the administration of acetylcysteine.[4] It has also been shown to be hepatotoxic in glutathione depleted mice,[5] but direct evidence relating to man is not presently available.

What are the lessons that can be learnt from our experience? Firstly one can be aware that clove oil is potentially very toxic but that an infusion of acetylcysteine may have a profound effect on the outcome. Secondly, that packaging and labelling of clove oil and other potentially toxic essential oils needs to be urgently reviewed. If all these oils were sold in bottles that had child resistant tops and were in restricted flow bottles, for example, dropper bottles, then even if a child managed to remove the top he/she would only be able to drink a very small amount. Also, the bottles need to be clearly labelled to indicate that the contents, if ingested, can be very toxic and urgent medical help should be sought if ingestion occurs.

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5 Mizutani T, Satoh K, Nomura H. Hepatotoxicity of eugenol and related compounds in mice depleted of glutathione: structural requirements for toxic potency. *Res Commun Chem Pathol Pharmacol* 1991; **73:** 87–95.